A Case of Non-Traumatic Subgaleal Hematoma Effectively Treated with Endovascular Surgery

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Key words: non-traumatic subgaleal hematoma, endovascular surgery

Summary

Non-traumatic subgaleal hematoma is very rare. We present a case of refractory non-traumatic subgaleal hematoma occurring in a 15-year-old male patient. The patient was successfully treated by embolization of the superficial temporal artery. This therapeutic approach to refractory non-traumatic subgaleal hematoma is discussed.

Introduction

A subgaleal hematoma (SGH) is usually associated with head trauma. The galea in a newborn infant can be pulled in a vertical direction and slipped in a tangential direction when exposed to an external force because the scalp is thin, the subcutis and periosteum are delicate, and the connection between these tissues is fragile. Therefore, an injured vein is considered to be a subcutaneous hematoma, and the infant is easily susceptible to a SGH or subperiosteal hematoma [1]. A small number of cases have been described in older children, occasionally as a result of minor head trauma such as hair braiding or hair pulling [2,3,4]. Non-traumatic SGH is rare, but may occur due to a ruptured aneurysm, failure of arteriovenous malformation, arteriovenous fistula (AVF) of the scalp or coagulopathy.

This report describes a case of effective endovascular surgery for non-traumatic SGH that was intractable by conventional therapy.

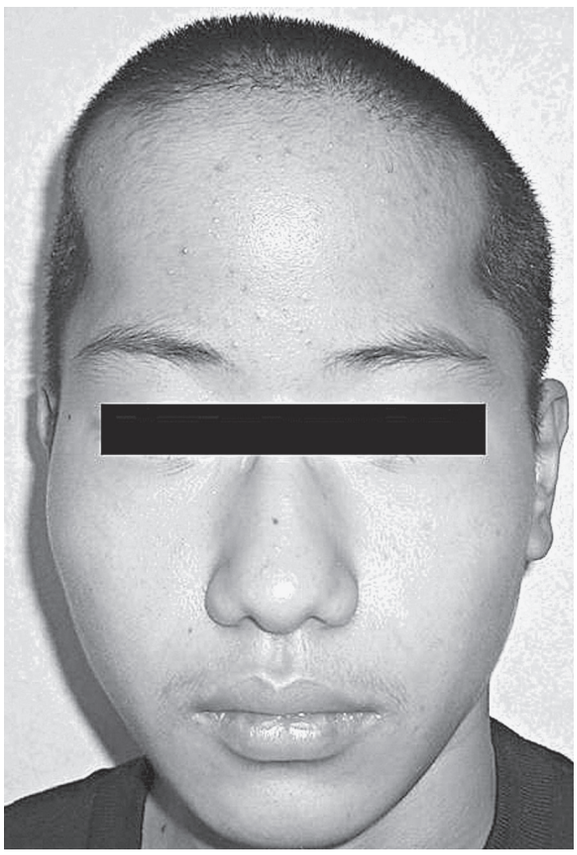
Case Report

A 15-year-old male experienced a sudden headache in the right frontal region on August 22, 2008. He came to the hospital on August 25 because the headache had increased in association with nausea and vomiting. The patient showed scalp swelling over the right cranium, but presented with no neurological deficits, and head computed tomography scanning revealed no abnormal findings. There was no history of trauma to the scalp. The patient came to the department of dermatology of the hospital on August 27.

An approximately 40 ml hematoma was aspirated and the scalp swelling disappeared. However, the swelling recurred, and the patient was referred to our department on August 29. The SGH was aspirated once more in our ward, and the scalp was fixed by compression with an elastic bandage on the patient's head. The absorbed liquid from the hematoma was fresh blood. The patient was hospitalized for examination on September 11 because the hematoma had recurred again.

Palpation showed a fluctuant, soft subcutaneous hematoma on the right frontal region. There was a subcutaneous hematoma across the midline and over the suture lines (Figure 1). These findings were thus considered to be characteristic of SGH. However, there were no traumatic findings. No neurological deficits were found.

The laboratory analysis of the patient's blood revealed normal blood coagulation, platelet ag-



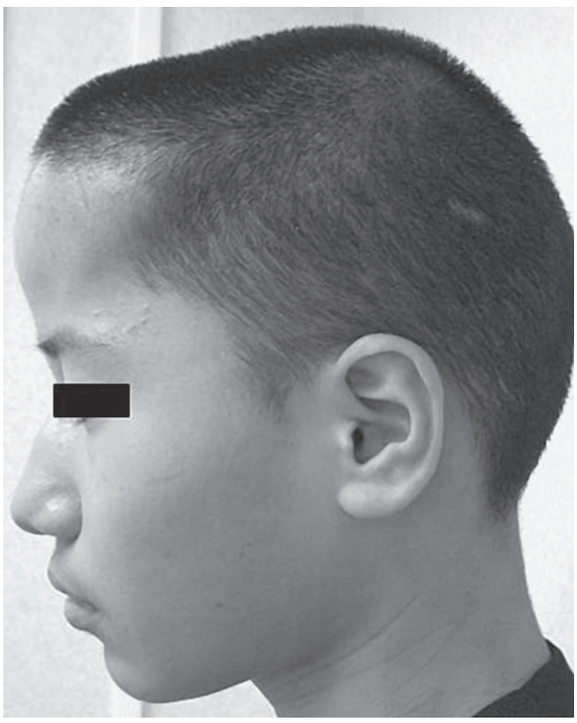
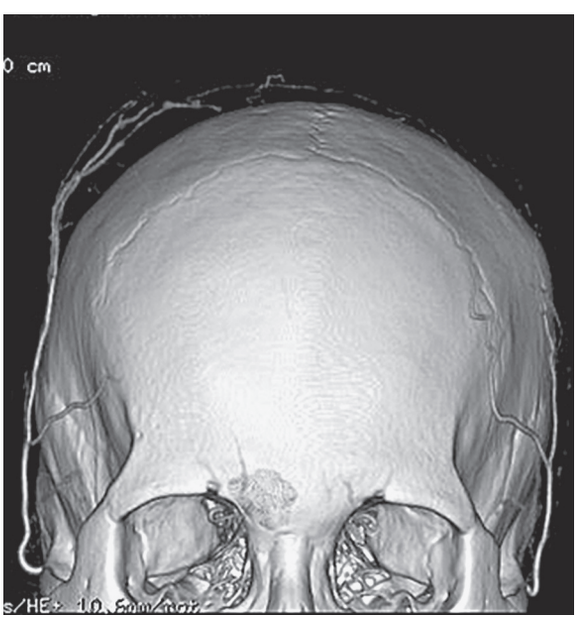


Figure 1 Photograph showing soft subcutaneous hematoma on the right side of the patient's head at the time of admission.

A



B

Figure 2 A,B) An initial computed tomography (CT) scan without contrast medium demonstrating a massive subgaleal hematoma (A). In addition, the right superficial temporal artery (STA) that developed is shown by pre-operative CT angiography (B).

gregation functions, and normal levels of blood coagulation factors.

Computed tomography (CT) revealed a large subcutaneous hematoma from the right frontal to the parietal region, but there was no abnormality on the skull or in the endocranial lesion. CT angiography (CTA) showed a right superficial temporal artery (STA) which developed more on the left than the right side, but no arteriovenous shunt was identified (Figure 2). Right extra-carotid angiography revealed a reticular shadow that was consistent with an SGH from a branch of the right STA (Figure 3). No microaneurysms were seen in the STA, and no abnormal shunts to a superior sagittal sinus such as AVF were identified. Internal-carotid angiography showed no abnormal findings. The initial treatment strategy was embolization of the bleeding vessel, followed by percutaneous hematoma aspiration and the application of a compression bandage.

Embolization was performed on September 24. The right STA was embolized because it showed reticular vessels with hematoma capsules and extravasation of the contrast medium into the hematoma. A guiding catheter (ENVOY 6Fr: Cordis/Johnson & Johnson, Miami, FL, USA) was introduced from the right femoral artery and advanced to the right external carotid artery. A microcatheter (Excelsior SL-10: Boston Scientific, Natick, MA, USA) was introduced into the distal portion of the right STA. Super selective angiography of the right STA immediately demonstrated a blush of contrast medium around the capsule of the SGH (Figure 4).

The AVF findings were not clearly identified at the time of the last angiography examination, but intraoperative angiography revealed a vein from the proximal portion of the hematoma at an early phase. We did not use liquid embolic material and particulate embolic material to avoid these materials migrating through the vein. Because the patient experienced a recurrence, we wanted to embolize the artery responsible. Therefore, embolization was performed using detachable coils (Serecyte: Micrus Corp., San Jose, CA, USA). Moreover, although no extravasation of contrast medium into the hematoma from a branch of the left occipital artery (OA) was observed, embolization was performed for the left OA because it contained reticular vessels with hematoma capsules. Angiography after embolization showed no hematoma capsule staining or leakage of contrast medium into the hematoma (Figure 5).

Percutaneous hematoma aspiration removed approximately 80 ml of fluid after the endovascular treatment, and a compression bandage was applied for three days. There has been no recurrence since the last hematoma aspiration at the one-year follow-up.

Discussion

Non-traumatic SGH is very rare. The cause of non-traumatic SGH is sometimes associated with aneurysms of the STA, scalp AVF, and coagulation disorders [5,6]. A differential diagnosis is necessary before treatment because the draining vein is often the venous sinus in the scalp AVF. Davis et al. reported on the diagnosis and management of neonatal subgaleal hemorrhage, and described it to be caused by a rupture of the emissary veins, which are connections between the dural sinuses and the scalp veins [7]. Kashino et al. reported that an angiographic examination of three out of four cases of atraumatic SGH showed a well-developed STA on the surface of the SGH, and one case had shown similar findings from the early stage of the development of the hematoma [8]. The authors suspected that this abnormal development of the STA was responsible for the massive SGH.

SGH is sometimes associated with coagulation disorders. For instance, Raffini et al. reported a von Willebrand disease case of SGH [9]. In the present patient, however, no such deficiency was identified, and the blood coagulation and platelet aggregation functions were normal. Therefore, coagulation disorders are not essential for the development of an SGH.

Because the patient had an STA with laterality, AVF was considered. Shinoda et al. reported that the most common symptom in traumatic AVF of the scalp was bruit (75.0%), the second a pulsatile mass (65.0%), and the third venous dilatation and pulsation (55.0%), and it is relatively easy to diagnose this disease in cases with the triad and a history of trauma [10]. Khodadad, in a review of 148 cases of scalp AVM, noted that half of the patients with congenital AVMs had red or purple birthmarks [11]. Komatsu et al. reported that the red birthmark and complex vascular network indicated that the lesion was a congenital scalp AVM [12]. In the present case, there were no characteristic symptoms or find-

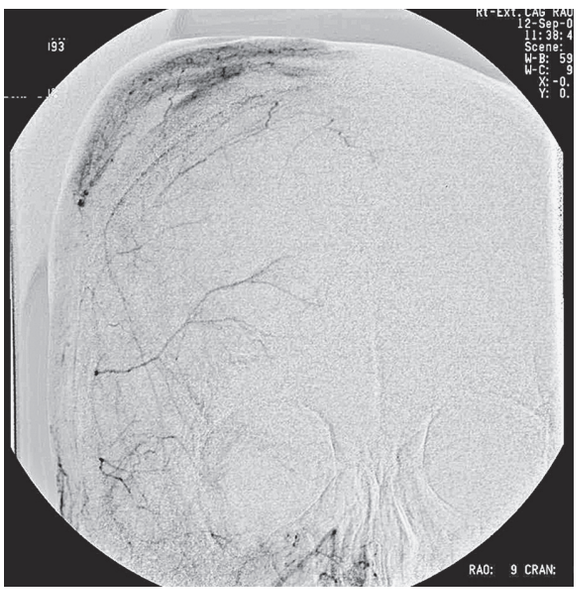


Figure 3 The right extra-carotid artery angiogram demonstrates extravasation of the contrast medium to the subgaleal hematoma.

Figure 4 A super-selective angiogram of the right STA demonstrates a stain of the hematoma membrane.

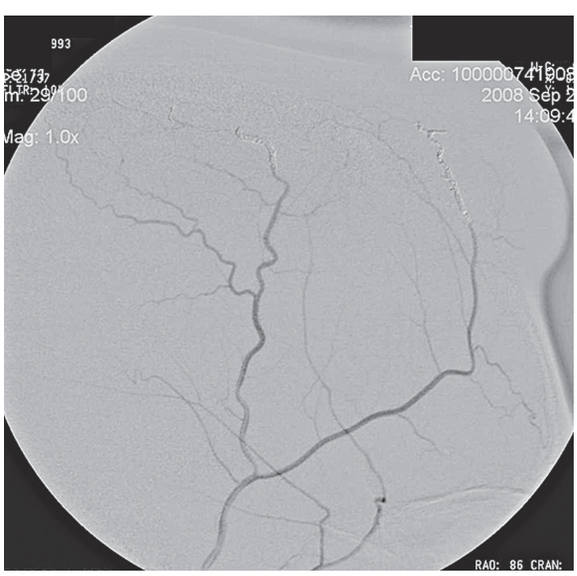


Figure 5 The post-embolization right extra-carotid artery angiogram demonstrates the complete disappearance of the hematoma stain and extravasation of the contrast medium to the subgaleal hematoma.

ings present that suggested scalp AVF. We therefore concluded that a cause of this subgaleal hematoma would not be scalp AVF. No definitive therapeutic strategy has yet been established for SGH. Therefore, there are various opinions concerning the treatment of hematoma. Falvo et al. elected surgical evaluation and pressure dressings to shorten the period of blood resorption and decrease the risk of infection, calcification, and blood reaccumulation [3]. Faber noted that aspiration may set the stage for infection or may be followed by recurrent bleeding and suggested that the extravasation itself may act as a tamponade to prevent further bleeding [13]. Beauchamp et al. noted that hematoma aspiration was unnecessary unless severe pain, impending necrosis of the overlying scalp, or evidence of infection was present [14]. The current patient had other accompanying symptoms, including headache and vomiting. A

pressure dressing was applied after two courses of puncture and aspiration, but had no effect. Achieving hemostasis by conventional therapy was unlikely, because angiography clearly indicated the extravasation of the contrast medium into the hematoma. The direct ligation of the STA directly was considered. The hematoma involved a large region of the head and a reticular artery developed from a contralateral OA, and therefore hemostasis by endovascular surgery was selected. There are several reports of the availability of endovascular surgery for the main source of bleeding [15,16,17]. Suzuki et al. reported that emergency embolization to manage bleeding from the MMA was a useful technique for patients with acute epidural hematomas [18]. This is the first reported case of successful endovascular treatment of an atraumatic SGH. Therefore, we referred to endovascular treatment methods for chronic subdural hematoma. The strategy of embolization varies with the cause of hematoma. Therefore the use of embolic material must be considered for endovascular surgery. No dangerous vascular anastomosis was found in the current patient, but the lesion was extensive. Therefore, the occlusion of proximal portions of the STA with platinum coils was selected as the optimal method. However, collateral circulation develops due to the occlusion of the proximal portions of the STA, and SGH must consider recurrent possibility. Bioactive coils (Serecyte: Micrus Corp., Sunjose, CA, USA) were used to optimize the embolic effect because recanalization may occur with embolization using bare platinum coils. The risks associated with endovascular treatment of STA include complications such as local inflammation, pain, migration of embolic material, and a perfusion disorder of the scalp. However, no complications occurred in the current patient. The first option for treatment of SGH is aspiration of the subgaleal blood and compression fixation. When angiography reveals the extravasation of contrast medium into the hematoma, then endovascular surgery may be an effective treatment choice.

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